PERIPHERAL NEURITIS IN ACUTE RHEUMATISM AND THE RELATION OF MUSCULAR ATROPHY TO AFFECTIONS OF THE JOINTS.

(Read before the Manchester Medical Society, 2nd May, 1888.)

BY

JUDSON S. BURY, M.D. (Lond.), M.R.C.P.,

Assistant Physician to the Clinical Hospital, and Medical Registrar to the Royal Infirmary, Manchester.

Reprinted from the "Medical Chronicle," June, 1888.

JOHN HEYWOOD,
Deansgate and Ridgefield, Manchester;
and 11, Paternoster Buildings,
LONDON.
1888.

PERIPHERAL NEURITIS IN ACUTE RHEUMATISM AND THE RELATION OF MUSCULAR ATROPHY TO AFFECTIONS OF THE JOINTS.*

THE main object of my paper to-night is to bring before your notice certain phenomena which are frequently met with during or subsequent to a genuine attack of articular rheumatism, and yet which are not described in text books on medicine, and are but imperfectly alluded to in special monographs on rheumatism. The phenomena are paralysis and atrophy of muscles, anæsthesia in the course of peripheral nerves, and occasionally, enlargement of the ends of bones. As atrophy of muscle is by far the commonest of these symptoms, and has long been noticed also in other affections of the joints, it will be more convenient first to consider the nature of this general relation, and then after a study and analysis of certain clinical features in rheumatic cases, we shall be in a position to discuss how far the element, common both to rheumatism and to other articular affections, whether injuries or diseases—viz., joint disturbance, is responsible in rheumatism for the wasting of muscles; or how far we may conclude, from any peculiar features of the atrophy, or from its association with other phenomena, that in rheumatism there are other factors at work equally potent with the joint irritation to set up wasting of muscle tissue.

The fact that any inflammation of a joint is almost invariably attended with rapid wasting of the muscles that move the joint, was first definitely described by John Hunter, in 1836. He recognised, too, that the atrophy is not the result of default of movement, for he says, when the other limb is examined, it is seen that the muscles there have preserved their volume almost entirely, although they have not been more moved than the muscles of the diseased limb. Hunter also noted that the muscles were sometimes paralysed, even when they were not atrophied.

* Read before the Manchester Medical Society, May 2, 1888.

In 1869, Ollivier, in his thesis on muscular atrophies, alludes to a case under the care of Duchenne, in which a traumatic synovitis of the knee was followed by great wasting of the thigh muscles, and he suggests the reflex nature of the phenomenon, and adds that such muscular atrophy is also met with in chronic articular rheumatism.

Vulpian also, in 1875, pointed out that the wasting begins almost as soon as the arthritis, and he ranges it amongst reflex atrophies. Fort, in 1876, showed that a slight arthritis of the shoulder may be accompanied by such rapid atrophy of the deltoid, that in eight days the flattening of the shoulder may simulate the deformity due to dislocation.

Sir James Paget, in his clinical lectures, also speaks of the extreme rapidity with which this muscular atrophy supervenes in all acute articular inflammations. But the most complete and suggestive account of the subject is to be found in a thesis by Valtat, published in 1877. He points out the importance of noticing that although it is difficult to separate paralysis from atrophy, yet sometimes the former is present without the latter; thus he mentions a severe sprain of the wrist where, after the pain and swelling had gone, there was found complete paralysis of the extensor muscles, while the atrophy was absent or doubtful.

The muscular atrophy, he says, is always marked and distinct at the beginning of the second week; thus, between eight and eleven days after a knee arthritis, the thigh on the affected side will measure about one inch less than the healthy thigh.

Pain is not essential as an antecedent, for an indolent hydrarthrosis may be followed by atrophy; and, conversely, Paget has observed similar wasting consecutive to a neuralgic joint pain without inflammation.

The extensor muscles are the most profoundly and the first to be affected. Thus, the quadriceps, when the knee is inflamed; if the hip, the glutei chiefly waste; if the shoulder, the deltoid; if the elbow, the triceps. But it is not uncommon, Valtat says, especially in old cases, to see other muscles affected, even an entire limb, although only one joint is inflamed.

Valtat obtained similar results by experimenting on dogs. He injected irritating substances, as ammonia, into the joints, and the resulting arthritis was always very intense, and the ensuing atrophy rapid and extensive, involving the whole limb in a few days; but, even here, the extensor muscles showed the greatest wasting.

Valtat, in seeking for an explanation, rejects disuse on account of the rapidity and degree of the wasting, and shows, too, that the effect cannot be due to any local inflammation of the muscles or nerves passing to them from the affected joints, for the whole length of a muscle suffers equally; moreover, he found no signs of inflammation on microscopical

examination of the wasted muscles, resulting from the traumatic arthritis produced by experiment, and he finally accepts Vulpian's reflex theory as the correct one, viz., that the irritation of the articular nerves so alters the nutrition of the ganglion cells of the cord as to seriously enfeeble the activity of the motor fibres derived from these cells.

Lastly, Charcot re-opens the question in clinical lectures delivered in 1883, and, while we acknowledge Valtat's thesis as the most complete account of the subject, and accord to Hunter the honour of the discovery, it is to Charcot, I think, that we feel particularly indebted for the dramatic force, and marvellous lucidity, with which he has set the picture before us. He relates the case of a man, aged 23, who, about a year prior to examination, struck his right knee in jumping over a fallen tree. The injury did not appear to be severe, for he walked several miles without difficulty, at length he stopped for a time, and then found himself unable to walk without a stick. For a week afterwards he kept his bed; the joint was swollen, though not very painful, but there was a remarkable loss of motor power in the limb. Two things were evident and striking, there was paresis and atrophy of all the muscles of the limb, but both paresis and atrophy were most marked in the quadriceps extensor. Tested by electricity, the muscles and nerves showed great quantitative diminution to both galvanic and faradic currents, but no qualitative change was obtained, showing that the condition was one of simple, and not degenerative, atrophy. Charcot obtained, however, lively muscular contractions by the application of the electric spark from the frictional machine. The knee-jerk was increased not only on the affected side, but on the other also. Charcot, after considering many hypotheses, finds himself obliged to admit the existence of a spinal affection, but in the absence of the reaction of degeneration, he cannot admit any serious change in the anterior horns of grey matter, and is reduced to the suggestion of a kind of stupor or inertia of the nerve cells. He explains the exalted knee-jerks, by supposing that while the cells of the affected region are in a state of torpor, there is a condition of increased reflex excitability in the rest of the cord. He relates also a case of chronic rheumatoid arthritis, affecting many joints, in which, although there was neither pronounced inflammation nor pain, there was rapid wasting, and loss of muscular power. There was the same loss of electric sensibility as in the other case, except that the right vastus externus gave the reaction of degeneration.

I may mention that there is now, in the Royal Infirmary, a patient under Dr. Simpson, who shows in the most typical manner the wasting of thigh muscles which so many authors have noted in affections of the knee. The patient is a man aged 20 years, the subject of subacute rheumatism. On admission, in addition to pain and slight swelling of

some of the joints, there was considerable effusion into the right knee; the thigh muscles felt distinctly smaller and softer than those of the left thigh, and when measured, two days later, or nine days from the time the knee first became painful, the right thigh was nearly one inch less in girth than the left one. The wasting appeared to affect all the muscles, and the adductors felt as soft as the quadriceps.

Charcot points out that in other cases of articular disease, spasmodic contraction, and not atrophy, is the dominant feature, as *e.g.*, in acute hip disease, and he refers to his description, given thirty years ago, of the deformities met with in rheumatoid arthritis, and he still looks on them as mainly produced by spasmodic contraction, developed by reflex action from the joint irritation. But when we see the deformities, he says the spasmodic contraction has usually ceased long before; the deformity, however persists, owing to the thickening of the tissues, the partial dislocations, and the shortening of the ligaments.

Now, with regard to this early spasmodic contracture in rheumatic cases, I am not quite sure that the evidence is satisfactory, or, at any rate, is not forthcoming in the large majority of cases. Thus, I think, one may follow out a case of acute rheumatism, and see a chronic deformity of the hand, for example, result without ever being able to detect a stage of contracture of muscle. Now, as in all such cases atrophy of certain muscles is met with, the deformity may, perhaps, be more correctly attributed to the *normal*, not spasmodic, unopposed action of healthy muscles, their antagonists being weakened and wasted; and, indeed, unless we see these deformities at a very late stage, it is usually pretty easy, so far as my experience goes, to reduce them; the joints of the fingers are generally freely movable, and can be put into normal positions. Nor am I satisfied that the reflex theory is an adequate explanation for the amount of atrophy often met with. Surely the stupor or inertia of the ganglionic cells cannot persist for months in a degree sufficient to produce such atrophy—say of the thigh, that the femur may be readily grasped, without any organic change in the nervous system. Dr. Ross has suggested to me that a depressed state or nutritional change of the central trophic cells may probably start degeneration, not of the anterior roots, but in the terminal fibres of the peripheric nerves, and Erb has advanced a similar hypothesis. I may mention, too, that Moussous, who has repeated the experiments of Valtat, and has then subjected the muscles and terminal nerves to a careful microscopical examination, has found in a few cases distinct degeneration of nerve fibres, but it must be confessed that the change was inconstant, and appeared to bear no proportion to the degree of muscular atrophy.

The subject seems one worthy of re-investigation, for, while we may

admit the reflex produced torpor of the ganglionic cells as a reasonable explanation of the sudden onset of the weakness and atrophy, it does not seem adequate to account for the progressive muscular atrophy which goes on long after the joint has completely recovered ; it is difficult to believe that such persistent symptoms can depend on any mere functional derangement. We know little of the pathology of the terminal axis cylinders, with their motor end plates ; possibly, in the future, it may be discovered that degeneration of these nerve elements may be responsible for many general atrophies now thought to be primarily and entirely disease of muscle tissue, or cases of so-called "simple muscular atrophy."

In such a proposed investigation, several remarkable clinical facts should be remembered.

(1) Charcot's description in his last volume of muscular atrophies in hysterical paralysis, whose onset and retrocession are equally rapid, and the general features of which are identical with the muscular atrophy met with in joint lesions—a simple muscular atrophy, Charcot says, due to a purely dynamic effect on the cord. And Dr. Gowers mentions in his new book that spinal sclerosis may develop from hysterical contracture, an almost unique example, he says, of structural change from functional disturbance. It is indeed curious to read of such structural changes in hysteria, a condition which we have always looked on as the most perfect type of a functional malady.

(2) In the same volume, a case of hemiplegia of organic origin with atrophy of the paralysed side is recorded. Descending degeneration was found, but the anterior cornua and motor nerves were quite healthy. Here, Charcot says, we can be certain that the atrophy depends on the central nervous system, hence the anterior horns, the trophic centres for the muscles, as they are not organically, must be dynamically altered.

(3) Cases of muscular atrophy from over use. Do they depend on purely local changes, or are they due to failure of the related ganglion cells of the spinal cord?

All these examples appear to be closely related in their pathology to that of arthritic muscular atrophy.

Having now cleared the ground by a consideration of what is common to all joint affections, it is time to turn to the muscular atrophies that cluster round a rheumatic attack, and which, by their distribution and associations, appear to me to have, at least very frequently, a special pathology.

Before relating illustrative cases, I must express my best thanks to the physicians of the Royal Infirmary for their kindness in allowing me to make use of their cases, and especially to Dr. Dreschfeld for the plaster cast of the hand of one of his patients, and for much help in

the examination of chronic rheumatic cases at the Crumpsall Hospital, and I am also indebted to Mr. Hardie, and to Dr. Barnes, one of the medical officers to the Crumpsall Hospital.

Case 1.—Kate A., 23 years, admitted under Dr. Dreschfeld, March 1, 1888. Her illness began early in January with pain and stiffness in the joints of the lower limbs. In February, last year, she had a similar, though less severe, attack of rheumatism. Prior to this she had fairly good health. A sister died æt. 27 years, of rheumatic fever and heart disease; her other sisters and brothers and her parents are quite healthy. On admission she was pale, sweating profusely, and had much pain in the limbs. In the lower limbs there was effusion into both knee joints; the ankles were very tender and slightly swollen. There was also tenderness in pressing over the external popliteal and posterior tibial nerves, together with some general hyperæsthesia of the skin and muscles. The feet were unduly extended at the ankle and there was a slight

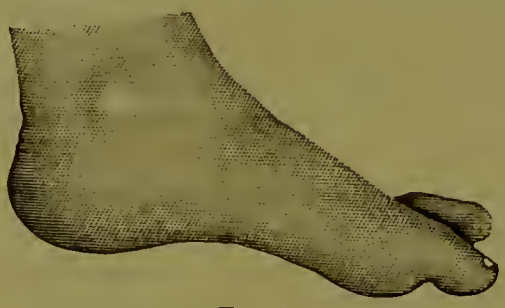

Fig. 1.

droop of the great toes, the other toes of the right foot were natural in appearance and position, but those of the left foot were hyper-extended at the metatarso-phalangeal joints and flexed at the other phalangeal joints, giving a somewhat claw-like appearance to the foot, and the dorsum of this foot, near the toes, was sunk, as compared with the right foot, and the muscles of the left leg were softer and more wasted than those of the right leg. On testing the cutaneous sensibility with pin prick and finger touch, distinct, though not absolute, anæsthesia was present on the inner side of the right great toe and foot and over the inner half of the sole, and it extended up on the inner side of the leg for about halfway to the knee, the area corresponding roughly to the distribution of the internal saphenous nerve.

As to the upper limbs, the right elbow was semi-flexed, stiff, painful on movement, and sore to palpation, especially in the ulnar groove, where around the nerve a gelatinous sort of swelling was felt. The hand was midway between supination and pronation, and movement in either of these directions was very limited. There was a slight swelling on the back of the right wrist. The knuckles were sore and

slightly swollen. The fingers were flexed at the metacarpo-phalangeal joints, and the patient could not extend them; there were depressions between the metacarpal bones on the back of the hand, owing to wasting of the interossei, the grooving being especially marked in the fourth space and in the first, where the muscular mass between the metacarpal bones of the thumb and index finger felt soft and thin. The hypothenar eminence was also wasted. She could not oppose the thumb against the tip of the little finger, and ad- and ab-duction of the fingers were feebly performed. There was a slight diminution of sensibility over parts of the right hand. The left arm and hand were affected in a similar manner. Well-marked tremor of the hands and fingers was observed when they were held out, and occasionally twitching of the

Fig. 2.

forearm muscles was seen. The tendon reactions in the arms and legs were increased. The heart and other organs were normal.

Two days later the partial anæsthesia in right leg and hand had disappeared.

On March 15th a slight systolic apex murmur was heard for the first time.

On April 2nd a crop of subcutaneous nodules was found over the external condyle of the left humerus, varying in size from a pin's head to a hempseed; also one on the knuckles of the index and middle fingers of both hands. These nodules were very painful on pressure. There was still much joint pain and sweating, and the tendon reactions were again found to be increased. The atrophy of the interossei, greater than formerly, was especially noticeable in the 1st, 3rd, and 4th spaces. The

little fingers were more flexed at the two terminal joints, and still hyperextended at the first. The forearm extensors appeared to be wasted. Both ulnar nerves were tender to pressure. On testing the interossei with electricity, they showed diminished reactions to both the faradic and galvanic currents, but probably no qualitative change to galvanism.

The patient is now (May 1st) in much the same condition. The nodules at left elbow are, however, much larger. It has been noticed for a week or two that the lower ends of both ulnar bones are distinctly thickened near the styloid process.

In this patient then, suffering from an attack of acute rheumatism, as proved by the articular affection, the sweating, the pyrexia—varying from 99° to 102°—the crops of subcutaneous nodules and the existence in all probability of endocarditis, we have (1) tenderness and swelling over the ulnar nerves with atrophy and paresis of the abductor indicis and other interosseous muscles supplied by these nerves; (2) probably similar atrophy of the interossei of the left foot supplied by branches from the posterior tibial; (3) temporary anæsthesia in the region of the internal saphenous nerve.

It would be tedious to relate other cases in detail, so perhaps you will take my word for it, that the following were truly examples of what is understood as articular rheumatism.

Case 2.—Another patient of Dr. Dreschfeld's, who had a severe attack of acute rheumatism accompanied by endocarditis, pericarditis, and pleuro-pneumonia, also presented marked wasting of the interossei. The hands were good examples of what might be called the typical rheumatic hand. Thus, when at rest, the metacarpal portion looks concave, the knuckles form a prominent anterior barrier of this shallow hollow, and the wrist the gradually sloped posterior boundary. The general concavity is interrupted (in this patient) by the extensor tendons of the middle and ring fingers. The concave metacarpus is well seen by looking at it from the ulnar side; then, instead of a straight or slightly convex line as in health, a concave one is seen, which is especially marked over the first space, owing to atrophy of the abductor indicis.

In this patient there was also slight thickening of the left ulnar nerve. There was distinct wasting of the muscles on the front of the legs, and purpuric spots were present on the outer sides.

Case 3.—A lady, under my own care, with rheumatic fever, which began in December, 1886, and was very severe and complicated by pericarditis and left pleurisy. Almost at the outset she had partial anæsthesia in the distribution of the ulnar nerve on one side, and also along the inner side of the same arm, corresponding to the distribution of the internal cutaneous nerve: this anæsthesia lasted three or four days. There was wasting of all the thumb muscles and of the interossei; and the

terminal phalanges of the fingers were slightly *hyperextended;* this made me think that the deep flexor of the fingers was affected, which is supplied by both median and ulnar nerves, and certainly there was evidence in the thumb and interossei muscles that both nerves were affected.

Case 4.—Another patient of mine, a man aged 70, had severe erysipelas which started in the right leg, and subsequently attacked the face. During convalescence he had what looked like an attack of rheumatism; the left elbow became very swollen and painful, and thickening round the ulnar nerve could be felt. All the muscles of the forearm and hand began to waste very rapidly, and showed well-marked fibrillary tremors, the fibrillation of the extensor muscles being extreme, and in a few days atrophy of these muscles was advanced. A little later the right elbow and forearm were affected in precisely the same way. It looked as if the rheumatic swelling about the elbow joint had pressed directly on the median, musculo-spiral, and ulnar nerves; or as if there were a separate inflammation of the sheaths of these nerves.

Case 5.—The following are notes of a man, lately under the care of Dr. Simpson, suffering from subacute rheumatism. Both forearms exhibit fibrillary tremors of the flexor and extensor muscles. The grasp is weak, especially of the left hand; all the muscles of the *right* upper are weak, the strength of the left being in marked contrast to them; this is especially noticeable in the triceps and supinator longus. Thickening is to be felt around the right ulnar nerve, and the interossei are decidedly atrophied. All movements of the fingers are weak and performed with much tremor, and opposition of the thumb is impossible, nor can the hand be completely closed. In the left leg there is evident diminution to cutaneous sensibility in the area supplied by the internal saphenous nerve. The external popliteal, while distinctly thickened on the right side, feels normal on the left. The feet show hyperextension of the toes at the first and flexion at the terminal phalangeal joints, and he cannot, by a strong voluntary effort, flex the proximal joints. All the tendon reactions are slightly increased. The patient remained in the hospital one month, then went to Cheadle. I saw him on his return in a few weeks, and though the hands were plumper, there was still distinct grooving between the metacarpal bones. Here then we have paralysis, atrophy, and fibrillation of muscles supplied by the musculo-spiral, median and ulnar nerves, together with anæsthesia of the skin, supplied by one internal saphenous nerve.

Case 6.—Another female patient under Dr. Dreschfeld had the chronic deformity of the hand seen in the woodcut, and which dated from an attack of rheumatic fever two years ago. When in the hospital she had pain and slight swelling in several joints, and her temperature

was occasionally raised. In this case there were several curious disturbances of cutaneous sensation; thus an area of well marked though not absolute anæsthesia over the ball of the right great toe, another extending along the outer side of the left leg from the malleolus half way up the fibula, and there was much hyperæsthesia of the soles of the feet and paræsthesia over the plantar surface of the toes of the left foot. In the right upper there was complete anæsthesia over the palmar and dorsal surface of the hand and fingers, partial anæsthesia over the thumb and the inner two-thirds of the anterior aspect, and over the back of the right forearm. The cutaneous sensibility was also slightly diminished over the upper arm. The anæsthesia varied in intensity, and was of temporary duration. The muscles of the right upper were all much weaker than those of the left upper.

In this case there was a suspicion that the anæsthesia was a hysterical phenomenon, but it was not so profound or so abruptly limited as is usual in that variety.

Fig. 3.

Another rheumatic case presented slight anæsthesia of the thumb and first finger. In another, a subacute case, there was partial ulnar anæsthesia and diminished sensibility also of the skin on the outer side of the left leg.

In most of the preceding cases the spine was examined and found to be free from tenderness; but there is another group of cases which I believe to be far from uncommon, in which, with certain parts of the spine tender to pressure, there is evidence of pressure on or irritation of the roots of some of the spinal nerves. The difficulty in such cases is to be sure that the presumed external pachymeningitis is secondary to a true rheumatism, and not primary, with associated joint pains from the neuritis. I will mention two :—

The first (*Case 7*), a man, æt. 40, under my care early this year, gave a history of chronic rheumatic joint pains. When I saw him he had no joint affection beyond some stiffness in the joints of the lower limbs, but there was a crop of subcutaneous nodules around the left patella. He had severe pain across the lower part of his back, and shooting pains down the sciatics, together with much tenderness over the sciatic and

other nerves in the legs. There was also much aching along the right arm, and I found the cutaneous sensibility markedly impaired along the inner side of the little, ring, and middle fingers, and the inner side of the palm, forearm, and arm; there was also some numbness over the supra-scapular fossa, and hyperæsthesia of the seventh cervical and first dorsal spines. Two days later the anæsthesia had gone.

Judging from his rheumatic history, and from the presence of rheumatic nodules, I thought it not unreasonable to suppose that the rheumatic poison had set up a slight inflammation of the dura mater, and possibly of the sheaths of the eighth cervical and first dorsal nerves.

Case 8.—The second, a man, 42 years, now in the Infirmary, under Dr. Dreschfeld. He had rheumatic fever two years ago, and has never been free from pain since. He was an in-patient for the first time last August, and then his ankles were swollen, and he had much pain down the spine. The muscles of the right upper arm were very weak, and the grasp of the right hand was extremely feeble. In September it was noted that he had severe pains in the knees, ankles, and great toes, but from the first the most severe pain has been down the right arm. At the present time all the movements of the right upper limb are weak, although the limb is as large and muscular-looking as the left one. There is slight numbness along the radial border of the right forearm. The spine is tender about the sixth and seventh cervical vertebræ, and to a less degree lower down, and the cutaneous tissues are sore to pinching from the spine radiating towards the shoulders. There is distinct thickening about the right ulnar nerve, and probably also of the right median nerve. Both hands and feet exhibit the condition known as erythromelalgia, a subject Dr. Morgan brought before this society at its last meeting (and I may add here that a slight degree of erythromelalgia of the feet is not a very rare phenomenon in articular rheumatism).

The tender spines, with hyperæsthetic radiating tracks to the shoulders, together with weakness of the right upper, point to pressure on nerve roots; and the thickening of the median and ulnar nerves, with the condition of the hands and feet, to a neuritis; and I think we have fair grounds for looking on the case as a rheumatic one.

A third, but rarer, group of cases are of great interest, suggesting, as they do, that a general progressive muscular atrophy occasionally starts from a genuine attack of articular rheumatism. This is an old notion; for example, Sir William Roberts, in his book on "Wasting Palsy," mentions a thesis by Thouvenet, who strangely enough contends that progressive muscular atrophy is primarily located in the peripheral nerves, and that it must be classed with rheumatic affections.

Case 9. Several years ago I saw an example of this in Pendleton—a woman who had a severe attack of rheumatic fever, which laid her up

fifteen weeks, and was followed by general muscular wasting. Dr. Reynolds, who was then doing duty at the Pendleton Dispensary, took notes of her case. I remember seeing her much wasted, with considerable articular deformity, and the right hand presented a marked "claw like" position.

Case 10.—A man, aged 44, recently under the care of Dr. Morgan, who had had several attacks of rheumatism during the last 20 years. Last October he was feverish, sweated much, and many joints were painful and swollen, and the doctor in attendance said he had rheumatic fever. He began to get thin afterwards, and has wasted ever since. There is now (January, 1888,) general and great muscular atrophy and fibrillation of many muscles. There is also some irregularly distributed anæsthesia on the right forearm, and the front of the right leg. All the tendon reactions are slightly increased. A mitral systolic murmur is heard, and is conducted round to the angle of the scapula. There is very evident enlargement of the ends of some of the long bones; thus, at the elbow, the bones feel much thickened, and this is especially noticeable with the head of the radius. Down each side of the front of the chest, and corresponding in appearance and position to the rickety beads met with in a young child, there is a remarkable row of bony nodules, apparently due to thickening of the ribs. These little knobs are certainly not the relics of rickety beads; the latter are due mainly to overgrowth of cartilage, and never persist into adult life.

In three or four other cases I have noticed hypertrophy of the ends of the long bones in acute rheumatism; sometimes the coronoid process of the ulna is felt thickened; sometimes its lower end near the styloid process, as in Case 1, and in the case just related, many bones were affected. There is sometimes great tenderness over the thickened portion of the bone, suggesting periostitis, and sometimes a little fibrous nodule may be felt adherent to the presumably-inflamed periosteum. [Such features as muscular atrophy, hypertrophy of bone, together with a similar deformity of hand both in rheumatic fever, and in the most chronic form of rheumatoid arthritis, point surely to an almost identical pathology.]

With regard to the cases of general muscular atrophy, are we to regard them as examples of a general trophic change set up reflexly by much joint irritation, or have we, as the anæsthesia in one case would seem to indicate, a degeneration of the extremities of a large number of peripheral nerves? The last group to which I would invite your attention, is that embracing local palsies and atrophies of muscle in cases of chorea.

Case 11.—There is a patient now in the Infirmary, under Dr. Simpson, a girl aged 20 years, who had articular rheumatism, affecting mainly the joints of the lower limbs, just before Christmas. In February

twitching began. On admission (March 2) chorea was severe and general. A week after admission a tendency to wrist-drop on both sides was noted, and the left ulnar nerve was distinctly thickened. A fortnight later the muscles on the back of the right forearm were greatly atrophied; the interossei, thenar, and hypothenar eminences were also wasted. You see in the woodcut the flattening of the forearm and the grooving on the back of the hand. Although the girl is generally very thin and wasted, the contrast between the right and left forearm is striking. The wasted muscles show diminished contraction to faradism, and an increased irritability to galvanism, but I could not satisfy myself as to a qualitative alteration.

In this case we have the following sequence: articular rheumatism, chorea, paralysis, and atrophy of muscles supplied by the musculo-spiral and ulnar nerves; and it is important to note that, so far as can be ascertained, the joints of the upper limbs were never painful or swollen.

Dr. Railton showed a little girl at a meeting of this Society, who, after an attack of chorea, developed paralysis and wasting of the lower

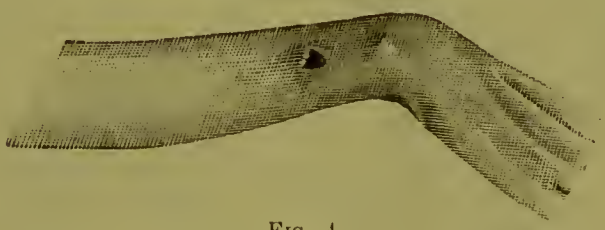

Fig. 4.

limbs, some of the muscles giving the reaction of degeneration. The knee-jerks were absent, and there were some slight sensory phenomena; and both Dr. Railton and Dr. Ross thought that the symptoms were best explained by a peripheral neuritis.

A very remarkable case was described by Dr. Bernard, of Londonderry, at the Belfast meeting of the British Medical Association, for the drawing and short description of which I am indebted to Dr. Thomas Barlow, of London. A child with chorea had wasting of the thenar and hypothenar muscles, and also of the interossei, and one hand presented a "main en griffe" attitude. Dr. Barlow thought that the chorea was attended or preceded by rheumatism, which had led to a neuritis of the ulnar nerve.

At a meeting of the Clinical Society, London, in 1884, Dr. Haddon showed a patient who, after an attack of acute rheumatism, showed trophic changes of the nails and the skin of the fingers, together with impaired sensation, also wasting of the muscles of the right forearm; and he mentioned two other cases where patches of anæsthesia were present in the arms and legs In the discussion which followed, my

friend, Dr. Thomas Barlow, described several cases of his own, short notes of which he has very kindly sent me, and which are of great importance, and as Dr. Barlow had referred to some of them in a previous but unpublished paper on rheumatism, read at Birmingham some years ago, he has the merit, I think, of first drawing attention to the subject. His cases are as follows:—

(1) A man with hyperpyrexial rheumatic fever, who during early convalescence had definite paralysis of one ulnar nerve and anæsthesia for a few days.

(2) Rheumatic fever, with bad pericarditis; during early convalescence definite paralysis of one ulnar and anæsthesia for a few days; also moderate wasting along the muscles supplied by the ulnar nerve to the hand. In this case there was also wasting of other limb muscles.

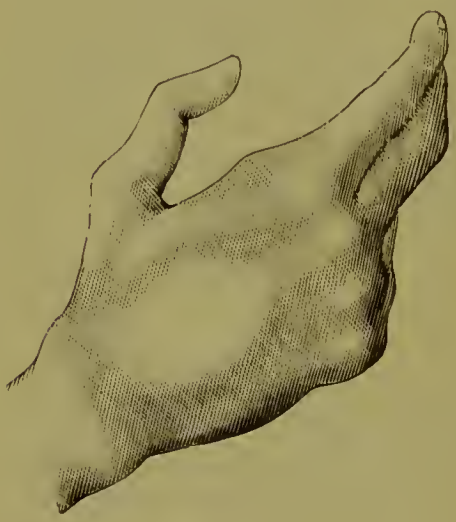

Fig. 5.

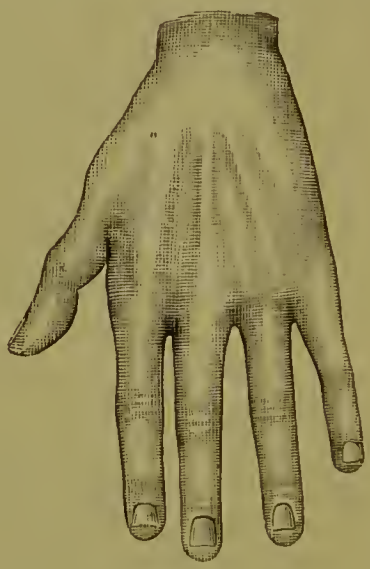

Fig. 6.

(3) A remarkable case of *extreme* wasting of all the interosseous spaces of the chest, following a very severe attack of rheumatic fever, but without pleurisy to account for the wasting.

(4) Agnes T., in February, 1883, had a slight rheumatic attack, followed in two or three days by chorea, which lasted two months. In November she had another rheumatic attack, followed by chorea, lasting five months. Again, in August, 1884, she had chorea and intercurrent rheumatism for three months. In January, 1885, æt. eight years, she had chorea, subcutaneous nodules, and a gangliform swelling on the back of the right hand (see Fig. 5). There was also marked atrophy of the interossei (Figs. 5—7), but no anæsthesia.

(5) Extreme wasting of the deltoid and scapular muscles as sequel to subacute rheumatism associated with a stiff neck.

(6) A boy had slight rheumatic fever and endocarditis; he came to Dr. Barlow some time after with mitral stenosis, stiff neck, and moderate wasting of the muscles of the arm and forearm down the side on which the hand was inclined to. There was a slight deficiency in response to faradism, but no other sign of the reaction of degeneration, and no sensory disturbance.

These are the only cases that I can find which directly bear on the subject before us.

Barbillon, in a recent thesis on the state of the cutaneous sensibility in acute rheumatism, points out, as Drosdoff did before him, that the faradic sensibility of the skin is usually diminished or abolished in the neighbourhood of the affected joints, and sometimes the diminution may be observed over the whole of an affected limb. Also, that sensibility to

Fig. 7.

touch and pain is also often diminished, but to a less degree, and less constantly than the faradic sensibility. He states, also, that muscular weakness and atrophy are frequently associated with the partial anæsthesia, but he only alludes to this incidentally, and does not localise either weakness or anæsthesia to special nerve territories. In discussing the pathogenesis, however, he suggests the possibility in some cases of an affection of the peripheral nervous system. As regards chronic rheumatic arthritis, I must not forget to mention the important investigations of Pitres and Vaillard, who examined the peripheral nerves of three cases and found them diseased in all, and there was a close correspondence between the muscular atrophy and the degenerated nerves. Thus, in one case where the leg muscles were atrophied, the corresponding nerves were profoundly altered; in another case, where all the limb muscles were normal in colour and structure, the muscular branches of

tho nerves were healthy, and the writers suggest a constant connection between the neuritis and the trophic disorders.

Ord has also drawn attention to the muscular atrophy, and wasted glossy skin seen in chronic rheumatism, attributed by him, however, to a central cause.

In the hand here pictured the skin over the back of the fingers was thin and glossy, and over the first phalanges showed brownish pigmentation. There was also atrophy of the interossei and thickening of one ulnar nerve.

In another chronic rheumatic patient it is interesting to compare the position of the thumb (Fig. 9) with that in a case of amyotrophic lateral sclerosis (Fig. 10) recently under the care of Dr. Ross. In both, owing

Fig. 8.

to wasting of the opponens pollicis, there is over extension of the metacarpal bone: In the former case there is also adduction of the thumb and hyper-extension of its terminal phalanx; but in the latter case (Fig. 10), owing to atrophy of the adductor and inner portion of the short flexor, the thumb is not only drawn backwards, but also away from the fingers, while its terminal phalanx is acted on by the long flexor.

It is time for me to sum up the gist of my observations; it is this:—

(1) That in articular rheumatism we constantly meet with the muscular atrophy and paresis common to other joint affections, and that while the sudden onset of these symptoms appears to be best explained by a reflex mechanism, whereby irritation conveyed along sensory nerves from the joint to the cord inhibits in some way the functional activity of the motor cells in the anterior horns, the progressive character and

duration of the atrophy suggest organic changes either central or peripheral. The presence also of increased tendon reactions, sometimes of contractures, and the fact that very rarely a lateral sclerosis may start from an arthritic attack* indicate that we may have changes not only in the motor cells but also probably in the terminations of the pyramidal tract or in that network of nerve fibrillæ by which they are supposed to be connected with the ganglionic cells, changes possibly not detectable by the microscope, yet, in all probability, more than functional.

(2) That one of the plainest and commonest phenomena of acute, subacute, or chronic rheumatism is wasting of the interosseous muscles of the hand; and that while some cases may be explained in the same way as other arthritic atrophies, viz., reflexly from inflamed knuckles, there is sufficient evidence that in a large number of cases the atrophy is due to an ulnar neuritis. Thus, in ulnar paralysis the index and

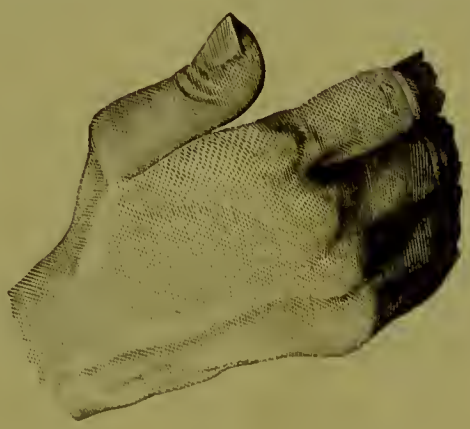

Fig. 9.

middle fingers are less affected than the two inner fingers, because, as you will remember, the outer two humbricales are supplied by the median. Now this is usually, though not invariably, the case in the rheumatic hand, as you see in Fig. 2, where the little and ring fingers show over-extension of the first phalanges and flexion of the others. In rheumatism too there appears to be a special incidence on the adductor pollicis and the abductor indicis. Confirmatory evidence of neuritis is afforded by the frequency with which impaired cutaneous sensibility is met with in the ulnar nerve territory, along with thickening and tenderness over the trunk of the nerve itself.

(3) That although the ulnar is by far the commonest nerve to be affected, there is substantial proof that other nerves of the brachial plexus and that branches of the lumbar and sacral plexuses are also frequently attacked.

* Gower's "Diseases of the Nervous System." Vol. I., p. 330.

(4) It is of great significance to note that these peripheral nerve symptoms may occur in a limb *quite free from joint irritation*, as, *e.g.*, during early convalescence from rheumatic fever. And if we thus often meet with paralysis, atrophy, or anæsthesia in the course of the ulnar nerve during an attack of rheumatism, or immediately after the pyrexia has subsided, in a limb whose joints are free, it appears to me that we have justifiable if not conclusive grounds for believing not only in a neuritis, but in one set up by the rheumatic poison. And when we read that Pitres and Vaillard, the more they examine cases of phthisis, tabes, and typhoid, the more constantly do they find evidence of neuritis in regions where, during life, symptoms of such neuritis were but slight, it is scarcely fanciful to admit the likelihood of a latent neuritis in rheumatism affecting a large number of nerves, and so accounting for much general muscular wasting.

Having referred these phenomena to the peripheral nerves, I must leave them, for it does not come within my province to discuss how far

Fig. 10.

these and other instances of peripheral neuritis may have a central origin; yet, with regard to this question, I may fitly conclude my paper by quoting words uttered by Graves, of Dublin, nearly forty years before the days of peripheral neuritis.

In his introductory remarks to "Diseases of the Nervous System," he says: "In considering the symptoms that accompany diseases of the nerves, pathologists have directed their attention almost exclusively to the nervous centres, and have looked on the brain, cerebellum, and spinal cord as the parts in which the causes of all nervous disorders reside, or in which they originate, forgetting that these causes may be also resident in the nervous cords themselves, or their extremities. In fact, gentlemen, pathologists have, with respect to the diseases of the nervous system, continued an error precisely similar to that which was so long prevalent with regard to diseases of the vascular system; for it is only lately that they have recognised the important truth that diseased vascular action may commence in the circumference." "And may not,"

he says in another passage, "the decay and withering of the nervous tree commence occasionally in its extreme branches? And may not a blighting influence affect the latter, while the main trunk remains sound and unharmed?"

BIBLIOGRAPHY.

I. References to Arthritic Muscular Atrophy.

John Hunter, 1836. Bonnet.—"Traité des maladies des articulations," 1845. Roux—*Annales de chirurgie*, 1845. Thouvenet.—"Thesis on muscular atrophy," 1851. Béziel.—"Thesis on muscular atrophy in relation to acute articular rheumatism," 1864. Ollivier (A.)—"Des atrophies muscularies," 1869, pp. 95-99. Here he refers to theses by Trastour, Plaisance, and Vergely, who as well as Charcot have described the muscular atrophy met with in chronic articular rheumatism. Sabourin.—"De l'atrophie musc. rhumatismale," 1873. Fort.—*Bull. et mem. de la Soc. de chir.*, 1872 and 1876. Paget.—"Clinical lectures and essays," 1875. Vulpian.—"Leçons sur l'App. vaso-moteur," 1875, t. ii.—Darde.—"Des atrophies conséc. à quelques affect. artic.," 1877.—Valtat. "Thèse," 1877. Vignes describes muscular atrophy in relation to joints in gout, rheumatism, and locomotor ataxy. "Thèse," 1880. Moudan.—"Thèse," 1882 (a good historical account of arthritic muscular atrophy). Cornillon.—"Amyotrophies consécutives à deux accès de goutte, simulant l'atrophie musculaire progressive."—*Le Prog. Medical*, No. 21, 1883. Méricamp.—"Thèse," 1883 (considers that syphilitic affections of the joints are accompanied by muscular atrophy). Petrone.—"Contrib. sull'atrofia musc. per artrite rheum. acuta. Lo Speriment Maggio," 1883. (Petrone regards the reflex theory as unsatisfactory.) Jacoby (G. W.). "The neurotic disturbances after joint affections."—*Journal of Nervous and Mental Dis.*, 2, 1884. [Jacoby says that *contractures* were not seen in the thirty cases examined, but that disturbances of sensibility and neuralgia were occasionally present.] Masse (E.).—"Des lésions du système musculaire dans les arthropathies," &c.—*Mém. de med. et chir.*, Paris, 1885, 113-131. Moussons (A. C.).—"Contrib. à l'étude des atrophies musculaires succedant aux affect. artic. ; recherches anat., path."—Bordeaux, 1885. Jubel Rémoy.—"Rhum. chronique fibreux amyotrophique à type rectiligne."—*Arch. génér. de Med.*, 1885. Charcot.—"Clin. lect. on senile and chronic diseases (New Syd. Soc., 1881), and "Maladies du syst. nerveux," Vol. III. 1887. Gowers.—"Diseases of the nervous system."—Vol. I., p. 381.

II.—References to Nerve Phenomena in Articular Rheumatism, &c.

Day (H.).—"The spinal origin of rheumatism."—*Med. Times*, 1867, p. 225, Jones (Handfield).—"Three cases of anomalous rheumatism."—*Med. Times*, 1873 Vol. 1. Drosdoff.—"Veränderungen der Sensibilität beim Gelinkrheum," &c.—*Centralbl. für die med. Wissensch.*, 1875, No. 17. Mora (J. L.).—"Des localisations spinales du rhum.," 1876. Valin.—"Cases of paralysis after rheumatism."—*Union med.*, 1878 (37, 38, 40). Kellner (W.).—"Ein Fall von rheum. Paraplege," Berlin, 1879. Descosse.—"Des troubles nerveux locaux consécutifs aux arthritis." Thèse, 1880. Boeck.—"A case of rheumatism with purpura and some anæsthesia of hands and legs, followed by atrophic paresis of arms and legs."—*Tidskr. f. pract. med.*, 1883, p. 257. Weber (L.).—"The neurotic origin of progressive arthritis deformans."—*New York Med. Rec.*, Nov. 17, 1883. Hutchinson.—"Gouty neuritis."—*Lancet*, 1884, Vol. II., p. 945. Jaccoud.—"Arthrite rhum. de la région cérvicale, compliquée de méningite ; paraplegie."—*Gaz. d. hop.*, 1884 (1170). Ollive—"Des paralysies chez les chorciques." Thesè, 1884. Ord.—*British Medical Journal*, 1884. Ross.—"Disease of nervous system," 1883, Vol. II., p. 1019 (cases by Landouzy, R. Hutchinson.

and Michel referred to). Renault (A.). relates the case of a young man, a rheumati subject, who was suddenly seized with signs of a myelitis in the lumbar region, followed in two days by pains and swelling in many joints, and later by peri- and endocarditis.—*Union Méd.*, 1884, Vol. IX. Gessler (H.).—"Motor end plates and their importance for peripheral paralysis." Leipzig, 1885. Hadden (W. B.).—"On certain nerve symptoms in rheumatic affections."—*Transactions, Clinical Society*, 1885, Vol. XVIII. Klippel.—"Des accidents nerveux der rhumatisme chronique et de la goutte."—*Annal. Méd. Chir. franc et étrang.* Paris, 1885, Vol. I. Remak (E.). "Ein Fall von generalisirter neuritis mit schweren electrischen Alterationcn, &c."—*Neurol. Centrbl.*, 1885, VI. Probably a generalised neuritis, or possibly a disseminated sclerosis which developed at the conclusion of articular rheumatism. Barbillon (L.). "De l'état de la sensibilité cutanée dans le rheumatisme articulaire aigu."—*Thesè de Paris*, 1886. Engesser. "Ueber einen Fall von Complication eines acuten Gebenkrhumatismus mit spinaler Lähmung."—*Arch. fur Psych.*, XVIII., 1886, S. 281. M., 57 years, had acute rheumatism with great tenderness over the lumbar spines; during convalescence atrophic paralysis of the lower limbs, no sensory phenomena. Engesser thinks probably the joints of the lumbar vertebrae were affected during the rheumatic attack, and that thus a meningomyelitis set up with especial incidence on the anterior horns. Recovery in a few months. Foxwell. "A case of rheumatic hyperpyrexia with nervous sequelae simulating multiple sclerosis.—*Lancet*, May 29, 1886. Frend. "A case of multiple neuritis, affecting the central and spinal nerves, probably of rheumatic origin, there being at the same time joint pains, much sweating and endocarditis."—*Neuro. Centralbl.*, 1886, p. 251. Garrod (A. E.).—"A contribution to the theory of the nervous origin of rheum.-arthritis."—*Lancet*, 1887, II., 1067. Also *Brit. Med. Journal*, 1888. I., 904. Girandeau (C).—"Des névrites périphériques."—(Rev. crit.)—*Arch. gén, de méd.*, 1887, II. A good summary of existing knowledge. *Pitres et Vaillard.* "Névrites périphériques dans le rhumatisme chronique."—*Rev. de Méd.*, No. 6, 1887. Railton (T. C.).—"Paralysis following chorea."—*Med. Chronicle*, Vol. V., p. 195.

John Heywood, Excelsior Printing and Bookbinding Works, Manchester.